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Disaggregating the Relative Influence of Genetic, Environmental and Individual Factors on LDL and HDL Cholesterols and BMI for a Sample of African American (AA) Mothers and Daughters

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DISAGGREGATING THE RELATIVE INFLUENCE OF GENETIC, ENVIRONMENTAL AND INDIVIDUAL FACTORS ON THE RELATIONSHIPS BETWEEN LDL AND HDL CHOLESTEROLS AND BMI FOR A SAMPLE OF AFRICAN AMERICAN MOTHERS AND THEIR DAUGHTERS.

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ABSTRACT

Brahler CJ, Harden JJ, McHone ML, Soules M, Evans E, Alyanak A, Diakpieng F, Vanderburgh P. Disaggregating The Relative Influence Of Genetic, Environmental And Individual Factors On The Relationships Between LDL And HDL Cholesterols And BMI For A Sample Of African American Mothers And Their Daughters. *JEPonline* 2006;9(1):24-31. There are many reports about the associations between blood lipids, body mass index (BMI) and dietary cholesterol intakes both within the individual and between related individuals. The purpose of this descriptive research project was to investigate the relationships between LDL and HDL cholesterols, body mass index and dietary cholesterol intakes for a sample of African American (AA) mothers and their daughters and to attempt to separate the contribution of genetic versus environmental factors. Mother and daughter participants (n =42 and 66, respectively) were 12-14-hours fasted when blood samples were drawn, heights and weights measured, and 24 hour food recalls completed. Mother (M) and daughter (D) LDL levels were significantly and positively correlated ($r=0.404$; $p<0.01$). Daughters' BMI (D-BMI) and HDL (D-HDL) levels were significantly and inversely correlated ($r=-0.36$ $p<0.01$). BMI and familial data should be used to identify children at high-risk for developing a negative blood lipid profile.

Key Words: Blood lipids, Body composition, Race, Women, Female

INTRODUCTION

Obesity is the most common health problem facing children in America today (1). Obesity can follow the child into adulthood and can be predictive of metabolic and cardiovascular diseases, such as diabetes, hypertension and hypercholesterolemia. Factors such as race, gender, age, heredity and socioeconomic class are associated with high cholesterol levels, and high cholesterol levels represent an independent risk factor for coronary heart disease (2). Parents strongly influence the health of their children through both genetic and environmental factors; however, research is deficient regarding the relative importance of each in determining a female child's LDL and HDL blood cholesterol levels.

Genetic and Environmental Influences on Obesity

Both genetic and environmental factors, such as metabolic and endocrine abnormalities, excess caloric intake and decreased physical activity, can be involved in the pathogenesis of obesity (3). Obesity is associated with diseases such as non-insulin-dependent diabetes mellitus (NIDDM), coronary artery disease (CAD), congestive heart failure (CHF), hypertension, hypercholesterolemia, certain cancers, and other medical problems (3). Studies of large populations show a positive linear relationship between BMI, morbidity and mortality (3). But obesity is also a serious health problem because of its association with diminished cardiovascular fitness and overall sense of well-being.

Some researchers have specifically focused on identifying conditions in children that are foretelling of obesity in adulthood. Serdula et al (1993) reported that 26-41 % of obese preschool, and 42-63 % school-age children become obese adults (4). According to Strauss, in 1999, 22 % of American children and adolescence were overweight and 11 % were obese (1), with maternal obesity being the number one predictor of childhood obesity (1). These statistics document the need for intervention. Epidemiologic literature published between 1970 and 1992 found that across all ages, the risk of adult obesity was at least twice as high for obese children than for non-obese children (4). Researchers appear to be in universal agreement that obese children are more likely to become obese adults.

African American Race and Obesity

Obesity is rapidly becoming the most prevalent health risk for children (5,6,7), and the problem is greater for African American children than for Caucasian children. African American girls are more likely to be overweight than Caucasian girls and African American children have recently been identified as a population at risk for developing obesity (8). The trend continues into adulthood (9), as 30 % of younger (18-30 years) and 77 % of older (45-65 years) black women are obese compared to 14 % and 46 %, respectively, for their white counterparts (10,11), and the prevalence of obesity is increasing faster in African American girls than in Caucasian girls (11). The overall health status of people of color (including African American women) is lower than that of the general American population (12,13). Sedentary lifestyle habits, poor dietary habits, and childhood obesity are on the rise (14,15,16), and women in minority groups are less physically active than the general American population. Physical inactivity seems to be influenced by a wide variety of psychological, social and environmental factors. Early adolescence is a developmental period during which important decisions are made regarding health behaviors. Attempts to instill healthy lifestyle habits during these years will likely be more successful than during adult years (5,17).

Genetic and Environmental influences on Blood Cholesterol Levels

The American Academy of Pediatrics recommends total cholesterol levels below 170 mg, and the LDL cholesterol levels below 110 mg. Common causes of high cholesterol include heredity, high-cholesterol diets, excess weight and lack of physical activity. Hypercholesterolemia may be monogenic or polygenic (18). Polygenic disorders, expressed in concert with environmental factors, result in the highest cholesterol levels in children (18). Strong familial aggregates are often found where environmental and genetic factors act in concert (18).

Blood Cholesterol Levels and Disease Conditions in Children

High blood cholesterol levels play a role in the development of premature coronary heart disease in adults, but no long-term studies have been conducted showing the relationship of blood cholesterol levels measured in childhood to coronary heart disease in later life. However that relationship can be inferred based on these facts:

1. Compared with their counterparts in other countries, US children have higher cholesterol levels and higher intakes of saturated fatty acids and cholesterol, and US adults have higher rates of coronary heart disease.
2. Autopsy studies demonstrate that early coronary atherosclerosis often begins in childhood and is related to high cholesterol.
3. Children and adolescents with elevated serum cholesterol levels, particularly LDL-cholesterol levels, often come from families with elevated values and in which there is a high incidence of coronary heart disease.

African American Race and Disease Conditions

African Americans have a higher prevalence of CHD and CHD mortality compared to the general population. High levels of obesity and low levels of physical activity play critical roles in inflating these statistics (19). On average, urban African American teenage girls have higher than average blood pressures, blood cholesterol levels, and BMI and reportedly consume foods high in saturated fat and sugar (20). The literature supports the need for health promotion and intervention among African American female children.

METHODS

Subjects

The subjects in this study included 108 African American mothers and their daughters (n = 42, 19 to 50 years old; and n = 66, 5 to 17 years old, respectively), residing in the Midwestern, urban city of Dayton, Ohio. Bulletins and announcements were used to recruit participants from local churches and a local community health and nutrition assistance program for low-income families. All subjects were informed that participation was voluntary and would have no effect on any services or benefits they might receive. Each subject received a \$25.00 stipend for participating. Transportation was provided to the study site if necessary.

Procedures

IRB Approval and Informed Consent Procedures:

The University of Dayton committee for the protection of human subjects approved this study and upon arrival, participants signed a University of Dayton Institutional Review Board approved informed consent to participate form. Mothers provided written informed consent to participate for themselves and their children, and children over the age of eight additionally gave assent. All participants were apparently healthy and medically stable.

Data Collection

Data collection took place at the University of Dayton Department of Health and Sport Science. Participants were 12-14-hr fasted when blood samples were drawn, heights and weights measured, and health history questionnaires and 24 hour food recalls completed. Blood samples were sent to a local medical diagnostics lab for analysis. Trained faculty and student technicians at the University of Dayton assisted the participants in reporting 24-hour food recall data, physical activity and health histories. Height and weight were measured. Body weight was measured in light clothing without shoes to the nearest pound on a beam balance scale, and height was measured without shoes with the head position in the Frankfort plane (eye and ear level) to the nearest 0.1 cm with an anthropometer. Weight was converted to kilograms to calculate BMI as weight in kilograms divided by height in meters squared. Subject age was recorded. Diet data were analyzed with the Nutritionist V computer program (March, 2000, First DataBank, Inc., San Bruno, CA). The software program was used only for determining absolute intakes.

Statistical Analyses

Data were categorized by BMI for normal weight (BMI<25), overweight (BMI 25-29.99), and obesity (BMI>30) and ANOVA tests run to determine differences in blood lipids between the groups. Correlation tests were run between mother and daughter average daily dietary cholesterol intakes (TC), low-density lipoproteins (LDL), high-density lipoproteins (HDL) and body mass index (BMI). Regression analyses were conducted to determine what variables were most predictive of daughters' LDL cholesterol levels, HDL cholesterol levels, and TC intakes. Alpha was set at 0.05.

RESULTS

Descriptive Data

Mothers' ages ranged from 21-50 years and the daughters from 5-17 years. The average age for the mothers (N=42) was 34.1 years and for the daughters (N=66) 10.03 years. Percentages of normal weight, overweight, and obesity for mothers and daughters were 18, 23, 59, and 59, 29, and 12, respectively (normal weight (BMI<25), overweight (BMI 25-29.99), and obesity (BMI>30) for adults and according to overweight and obesity cutoff points for children by age and sex as proposed by Cole et al and the International Obesity Task Force. Average LDL levels of 102.9 mg and 98.5 mg were low compared to the NIH cut off point for normal LDL of less than 130 mg. Average HDL levels were 53.9 mg and 54.1 mg for mothers or daughters, respectively compared to the NIH guideline 50 mg. See Table 1 for a summary of descriptive statistics.

Table 1. Descriptive data of the subjects.

Descriptives	Age (yrs)	BMI	LDL (mg)	HDL (mg)	WHR
<i>Mothers</i>					
Normal wt	35.1±7.1	20.8±3.0	100.0±36.4	59.2±12.2	0.8±0.1
Overweight	33.9±7.7	27.8±1.4	103.0±32.0	51.4±14.7	0.9±0.1
Obese	33.2±9.2	39.2±6.5	105.6±33.1	51.2±16.1	0.9±0.1
<i>Daughters</i>					
Normal wt	9.4±4.0	17.6±2.4	109.7±32.5	55.0±13.6	0.8±0.1
Overweight	11.3±2.6	22.6±2.8	97.3±28.9	60.5±12.3	0.8±0.1
Obese	10.4±4.3	27.5±7.1	88.3±30.2	46.7±17.2	0.9±0.0

Correlations

The child's LDL levels were significantly and positively correlated with maternal LDL levels (LDL $r=.404$; $p<0.01$). The daughters' HDL levels were significantly and negatively correlated with daughters' BMI ($r=-0.36$ $p<0.01$). The daily cholesterol intake levels for mothers and daughters were significantly correlated ($r = .447$, $p < 0.01$). Mother and daughter BMI scores were significantly and positively correlated ($r = 0.278$; $p < 0.05$). See Table 2 for a summary of correlation statistics.

Regression Analyses

Stepwise multiple regression analysis to predict the dependant variable LDL cholesterol levels for the daughters (LDL-D) using the independent variables LDL cholesterol levels for the mother (LDL-M) and BMI-D resulted in LDL-M being included in the regression analysis ($p=0.003$) and BMI-D being excluded in the regression ($p=0.59$).

Table 2. Summary of results from correlation analyses.

Correlations	R value	P value
LDL-M and LDL-D	0.404(**)	0.003
BMI-D and HDL-D	-0.360(**)	0.006
TC-D and TC-M	0.447(***)	0.0001
BMI-D and BMI-M	0.278(*)	0.024

* Correlation is significant at the 0.05 level (2-tailed).

** Correlation is significant at the 0.01 level (2-tailed).

*** Correlation is significant at the 0.01 level (2-tailed).

$$\text{EQUATION: LDL-D} = 65.1 + (0.344) \text{ LDL-M}$$

Stepwise regression analysis to predict the dependant variable total dietary cholesterol intake for the daughters (TC-D) using the independent variables total dietary cholesterol intake for the mothers (TC-M) and BMI for the daughters (BMI-D) resulted in TC-M being included in the regression analysis ($p=0.0001$) and BMI-D being excluded in the regression ($p=0.732$).

$$\text{EQUATION: TC-D} = 128.128 + 0.362 (\text{TC-M})$$

Stepwise regression analysis to predict the dependant variable HDL cholesterol levels for the daughters (HDL-D) using the independent variables TC total dietary cholesterol intake for the daughters (TC-D) and BMI-D resulted in BMI-D being included in the regression ($p=0.007$) and TC-D was excluded in the regression ($p=0.59$).

$$\text{EQUATION: HDL-D} = 76.444 + (-1.004) \text{ BMI-D}$$

ANOVA tests revealed there was not a significant difference for LDL levels between BMI groups (normal weight, overweight, and obesity) for daughters ($p = 0.487$) or mothers ($p = 0.966$). There was not a significant difference for HDL levels between BMI groups (normal weight, overweight, and obesity) for daughters ($p = 0.082$) or mothers ($p = 0.566$).

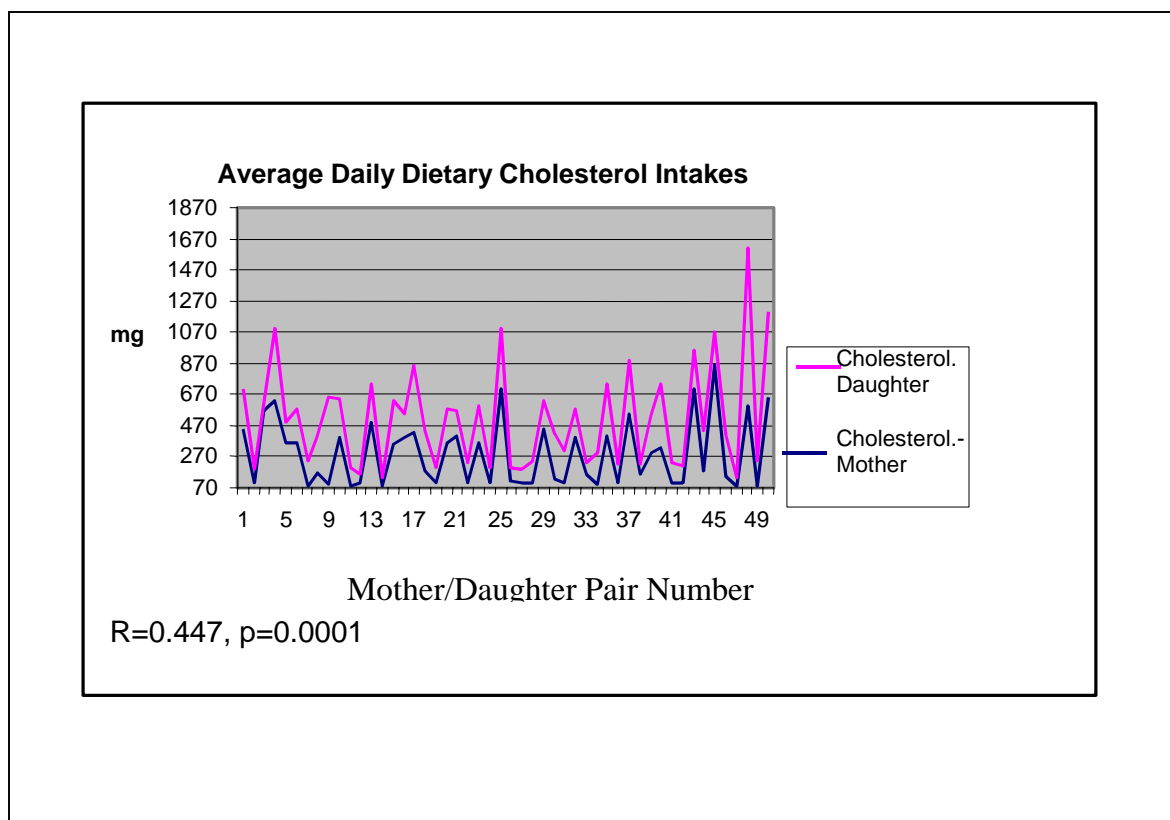


Figure 1. Average daily dietary cholesterol intakes for all daughters paired with their respective mothers.

Although not a conventional use of a line graph, Figure 1 provides a very visual depiction of the high correlation between average daily dietary cholesterol intakes for mothers and daughters (Figure 1).

This relationship is even more pronounced when only the mothers and daughters classified as overweight or obese are considered (Figure 2).

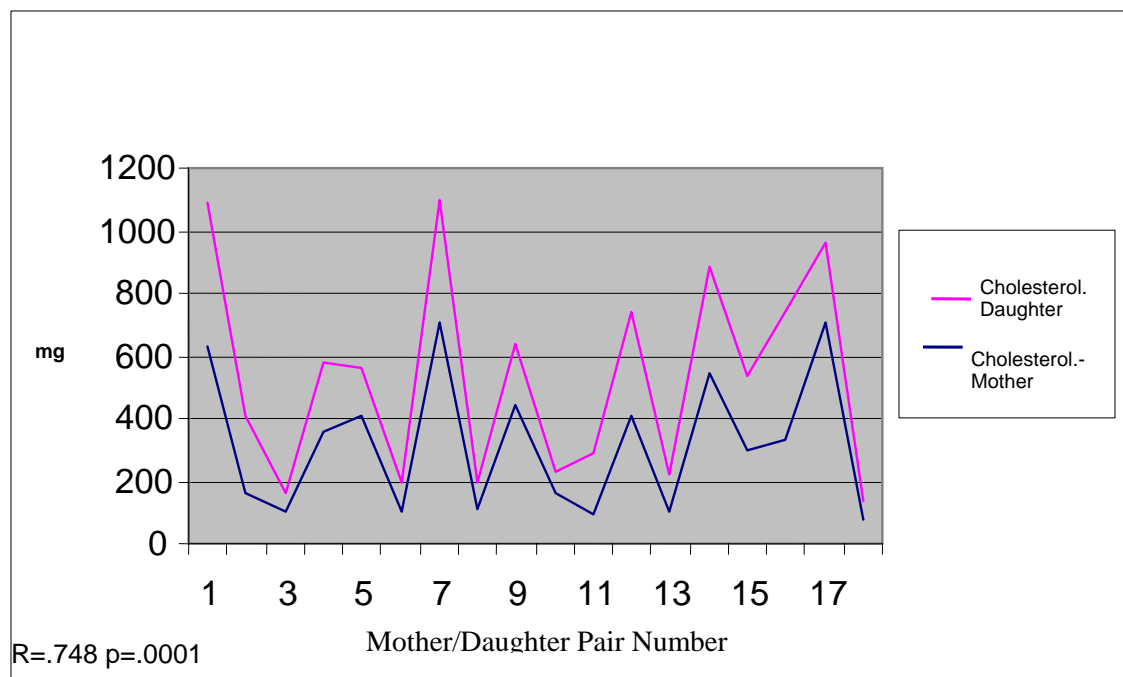


Figure 2. Average daily dietary cholesterol intakes for overweight and obese daughters paired with their respective mothers.

DISCUSSION

The findings of this study can be broken down into three main categories. Namely, these three categories are: genetic, environmental and one we have added called “individual”. We have attempted to interpret our results in such a way as to provide a comprehensive profile of the contribution of each factor to the LDL and HDL cholesterol levels for the AA daughters in our study sample. In order to do this, within each category we completed a statistical differential diagnosis (DfDx) of the effect of each on HDL and LDL levels.

Genetics

LDL levels in our sample of young AA females appear to be primarily inherited from their mothers as evidenced by the high correlation between mother and daughter LDL levels. Additionally, LDL levels were not significantly different between BMI groups (normal weight, overweight, obesity). This fact provides additional evidence that the genetic association is greater than the association with BMI independent of heredity. Regression analyses substantiated the genetic link because LDL levels for the daughters were best predicted by LDL levels for the mothers because stepwise regressions included LDL-M as the best predictor for LDL-D and omitted daughter’s own BMI as a predictor of LDL-D.

Individual

HDL levels seemed to be mostly dependent upon the young AA females’ own BMI values as evidenced by the facts that the girl’s individual BMI and HDL levels were significantly correlated but ANOVA tests revealed there was not a statistically significant difference in HDL levels between BMI groups (normal weight, overweight, obesity). Regression analyses substantiated the “individual” link because HDL levels for the daughters were best predicted by the daughters’ own BMI levels not the

mothers' HDL levels. Stepwise regressions included BMI-D as the best predictor for HDL-D and omitted LDL-M as a predictor of LDL-D.

Further, the HDL levels for mothers and daughters were not significantly correlated ruling out a significant genetic influence. We fully appreciate that BMI is a construct of both genetic and environmental influences and we use the term "individual" to distinguish that each daughter's individual BMI had the significant correlation with HDL. We wish to distinguish "individual" BMI from "categories of BMI (normal, overweight, obesity) because HDL levels were not significantly associated with these BMI categories.

Environmental

The evidence in this study for a strong environmental influence was not directly linked to blood chemistry values, but rather to negative dietary practices that may be associated with increasing LDL cholesterol levels. Namely, there was high correlation between average daily dietary cholesterol intakes for mothers and daughters. The correlation was highest between mothers and daughters classified as overweight or obese, not normal weight, suggesting that this detrimental environmental influence is perhaps strongest for those individuals in those BMI categories.

CONCLUSIONS

Given the fact that African American women are at high risk for developing cardiovascular and metabolic diseases, young African American females should be encouraged to decrease dietary cholesterol intakes and to reach and maintain normal body weights. Female children whose Mothers are known to have elevated levels of LDL cholesterol should be preferentially targeted for intervention. Children with obesity and whose mother has high LDL cholesterol levels are at double the risk for developing negative health consequences and perhaps should be selectively targeted for intervention.

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REFERENCES

1. Strauss RS, Knight JK. Influence of the Home Environment on the Development of Obesity in Children. *Pediatrics* [Online] 103:6, 1999 e85. Retrieved November 30, 1999 from <http://www.pediatrics.org/cgi/content>.
2. USDA Center for Nutrition Policy and Promotion (1999, May). *Profile of Overweight Children*. Nutrition Insights.
3. National Institute of Health (1985) Health Implications of Obesity: National Institutes of Health Consensus Development Conference Statement. Retrieved December 12, 2001 from <http://isis.nlm.nih.gov/nih/cdc/www49txt.html>
4. Serdula MK, Ivory D, Coates JR, Freedman DS, Williamson DF, Byers T. Do Obese Children Become Obese Adults: A Review of Literature: *Prev Med* 1993;22:166-167.

5. Armellini F, Robbi R, Zamboni M, Todesco T, Castelli S, Bosello O. Resting metabolic rate, body-fat distribution, and visceral fat in obese women. **Am. J. Clin. Nutr.** 1992;56:981-987.
6. Dietz WH. Risk factors for childhood obesity. **Int. J. Obesity Rel. Met. Disor.** Childhood Obesity Satellite Symposium of the 8th International Congress of Obesity. 1998;22(Suppl.4):S2.
7. Goudas M, Bidde S, Fox K, Underwood M. It ain't what you do, it's the way you do it! Teaching style affects children's motivation in track and field lessons. **Sport Psychology.** 1996;9: 254-264.
8. Dwyer JT, and CATCH Cooperative Research Team. Prevalence of marked overweight and obesity in a multiethnic pediatric population: Findings from the Child and adolescent trial for cardiovascular health (CATCH) study. **J. Am. Diet. Assoc.** 2000;100:1149-1156.
9. Williamson DF, Kahn HS, Beyers T. The 10-year incidence of obesity and major weight gain in black and white US women aged 30-55 years. **Am. J. Clin. Nutr.** 1991;53(Suppl.):S1515-S1518
10. Folsom AR, Burke GL, Byers CL, Hutchinson RG, Heiss G, Flack JM, Jacobs, Caan B. Implications of obesity for cardiovascular disease in blacks: The CARDIA and ARIC studies. **Am. J. Clin. Nutr.** 1991;53(Suppl.):S1604-S1611.
11. National Center for Health Statistics: NHANES III reference manuals and reports. Centers for Disease Control and Prevention: Hyattsville, MD, 1996.
12. Gordon-Larsen P, McMurray RG, Popkin BM. Determinants of physical activity and inactivity patterns. **Pediatrics** 2000;105:6.
13. Kriska A. Ethnic and cultural issues in assessing physical activity. **Res. Quart. Exerc. Sport.** 2000;71:47-53.
14. Bar-Or O. Juvenile Obesity, Physical Activity, and Lifestyle Changes: cornerstones for prevention and management. **The Physician and Sports Medicine** 2000;28(11): 51-58.
15. Golan M, Weizman A, Fainaru M. Impact of treatment for childhood obesity on parental risk factors for cardiovascular disease. **Prev. Med.** 1999;29:519-526.
16. Pidlich J, Pfeffel F, Zwiauer Z, Schmidinger H. (November 1997). The effect of weight reduction on surface electrocardiogram: a prospective trial in obese children and adolescents. **Int. J. Obesity Rel. Met. Disor.** 1997;21:1018-1023
17. Epstein LH, Saelens BE, Myers MD. Effects of decreasing sedentary behaviors on activity choice in obese children. **Health Psychol** 1997;16(2):107-113.
18. Klish WJ, Baker SS, Cochran W, Flores C, Georgieff M, Jacobson MS, Lake A. (1998, Jan). Cholesterol in Childhood. **Am Acad Ped.** Retrieved November 1, 2001 from Lexis-Nexis database site <http://web.lexis-nexis.com/universe>.
19. Potts JL, Thomas J. (1999, Mar). Traditional coronary risk factors in African Americans. **Am J Med Sci** (MEDLINE Identifier Number 99198792) Abstract retrieved December 12, 2001 from OhioLINK research database site <http://olc3.ohiolink.edu>.
20. Fardy PS, Azzolini A, Magel JR, White RE, Schmitz MK, Agin D, Clark LT, Bayne-Smith M, Tekverk L, (2000, Mar-April). Gender and ethnic differences in health behaviors and risk factors for coronary heart disease among urban teenagers: the PATH program. **J Genet Specif Med** (MEDLINE Identifier Number 21148932) Abstract retrieved December 12, 2001 from OhioLINK research database site <http://olc3.ohiolink.edu>.
21. John FA, Philip R, Lechard JT, Ashok V, James KY. Exercise induced hyper-calcemia and calcitropic hormones. **J Lab Clin Med** 1985;106:229-232.
22. Plowman S, Smith D. **Exercise Physiology for Health, Fitness, and Performance.** 2nd Edition. San Francisco: Benjamin Cummings, 2003.